Brazelton Neonatal Behavioral Assessment Scales and the Graham/Rosenblith neurological soft signs scale showed hypertonia, irritability, abnormal cry, and other neurological soft signs at 2, 15, and 30 days. Brainstem auditory evoked responses and clinical EEG were essentially normal at 20 days, 3, 6, and 12 months. EEG sleep pattern was fragmented at 20 days and 3 months, and abnormal respiratory patterns were noted to 6 months. Psychometric (Bayley Scales, Terman Merril and McCarthy Scales) and diagnostic testing (Fagan Test of Infant Intelligence) yielded scores within normal limits out to 3 years. At every examination, however, testing protocols noted some combination of restlessness, agitation, distractibility, high energy level, lack of persistence, short attention span, and poor fine motor control. The mother describes the child as very difficult, with low frustration tolerance.

Lead-glazed ceramic ware, common in Mexico, is widely used by Hispanics in the United States, who often bring it from Mexico.[1,2] Tourists import such items as gifts, and the number of recalls of commercially imported ceramic ware likely underestimates the quantity of leaded items available.[3]

Uninformed physicians can adversely affect treatment of lead poisoning. Toxic levels of lead produce symptoms that can be confused with other disorders. The pattern of hospital admission, unconfirmed diagnoses, reduction of symptoms when the patient is removed from the lead source during hospital stay, discharge, and re-exposure has been noted before.[4] Mothers with high lead levels expose their infants through maternal milk. Standardized psychometric tests are frequently without value in detecting damage from lead in children up to 3 years, even though behavioral disturbances are clear. □

Stephen J. Rothenberg, PhD
Lourdes Schnaas-Arrieta, BS
Juan Carlos Ugartechea, MD
Estela Perroni-Hernandez, BS
Irving A. Perez-Guerrero, BS
Selene Cansino-Prtiz, MS
Vicente Salinas, MD
Francisco Zea-Prado, MD
Aleksandra Chicz-Demet, PhD

Stephen J. Rothenberg is with the National Institute of Perinatology, Mexico City, and Fairview State Hospital, Costa Mesa, Calif. All other authors are also with the National Institute of Perinatology in Mexico City. Juan Carlos Ugartechea has since died. Requests for reprints should be sent to Stephen J. Rothenberg, PhD, National Institute of Perinatology, Department of Developmental Neurobiology, Montes Urales Sur 800, Lomas Virreyes, Mexico City, CP 11000, Mexico.

Acknowledgments

This study was supported in part by the Secretariat of Health, Mexico, Grant 1R01 MH41446, NIMH, USPHS, a grant from an anonymous donor, and financial assistance from Frandon Enterprises, Inc.

References

1. Trotter RT. The cultural parameters of lead poisoning. Presented at the National Institute of Environmental Health Sciences Meeting; January 1989; Research Triangle Park, NC.
2. Wallace DM, Kalman DA, Bird TD. Hazardous lead release from glazed dinnerware: a cautionary note. *Sci Total Environ.* 1985;44:289–292.
3. Committee on Energy and Commerce House of Representatives. *Lead in Housewares, Hearing. (Serial No. 100-134.)* Washington, DC: US Government Printing Office; 1988:50–53.
4. Bird TD, Wallace DM, Labbe RF. The porphyria, plumbism, pottery puzzle. *JAMA.* 1982;247:813–814.

Could Sunscreens Increase Melanoma Risk?

Topically applied chemical sunscreens prevent sunburn.[1] One of the most common sunscreens, para-aminobenzoic acid (PABA), was invented in 1922, and commercial products containing sunscreens became available in 1928.[2] High sun protection factor (SPF) sunscreens, largely based on PABA and its esters, became widely available by the late 1960s and early 1970s.[3,4] High SPF sunscreens have been widely recommended for the prevention of skin cancer, including melanoma.[5–7] It has been assumed that the action spectrum for initiation and promotion of melanoma and basal cell carcinoma is identical to that of sunburn.[8] Sunscreens have been strongly recommended for persons with fair coloring and those with a history of skin cancer,[9,10] and use of sunscreens has become widespread. A large proportion of adults in the United States report using sunscreens during recreation,[11] and the American Medical Association has recommended that frequent use of sunscreens should become a standard procedure for children.[6]

Although sunscreens, including PABA and its esters prevent sunburn,[1,2,12] there has never been any epidemiological or laboratory evidence that they prevent either melanoma or basal cell carcinoma in humans.

Worldwide, the countries where chemical sunscreens have been recommended and adopted have experienced the greatest rise in cutaneous malignant melanoma, with a contemporaneous rise in death rates. In the United States, Canada, Australia, and the Scandinavian countries, melanoma rates have risen steeply in recent decades, with the greatest increase occurring after the introduction of sunscreens.[13–17] Death rates in the United States from melanoma doubled in women and tripled in men between the 1950s and the 1990s.[18] The rise in melanoma has been unusually steep in Queensland, Australia, where sunscreens were earliest and most strongly promoted by the medical community.[19] Queensland now has the highest incidence rate of melanoma in the world.[20] In contrast, the rise in melanoma rates was notably delayed elsewhere in Australia,[20] where sunscreens were not promoted until more recently.

The SPF of sunscreens concerns solely their ability to absorb ultraviolet B (UV-B) light.[21] Even sunscreens with high SPF factors can be completely transparent to ultraviolet A (UV-A),[21] which includes 90% to 95% of ultraviolet light.[22] UV-A blocking ingredients, which have commonly been added to most sunscreens since 1989, block only half the UV-A spectrum and provide a protection factor against delayed UV-A induced erythema of only 1.7 at usual concentrations.[23]

Both UV-A and UV-B have been shown to mutate DNA and promote skin cancers in animals.[24,25] UV-A also penetrates deeper into the skin than UV-B.[26] Because of the energy distribution of sunlight[22] and filtering by the outermost layers of the skin,[26] melanocytes receive up to 70 photons of UV-A for every photon of UV-B.

While largely transparent to most of the UV-A spectrum, sunscreens effectively block UV-B. UV-B is the normal stimulus for accommodation of the skin to sun, such as thickening and increased pigmentation.[27] Sunscreens also inhibit the skin's production of vitamin D, which is similarly dependent on UV-B.[28] Laboratory findings indicate that vitamin D metabolites suppress growth of melanoma cells,[29] suggesting the possibility that vitamin D deficiency in the skin may have a role in the etiology of melanoma.

While few epidemiologic studies have examined the relationship of sunscreen use and skin cancer, two studies suggest that sunscreens may not be effective in preventing skin cancer. A large

case-control study showed higher risks of melanoma in men who used sunscreens,[30] and a large prospective study showed a higher incidence of basal cell carcinoma in women who used sunscreens.[31] The excess risks in the latter study persisted after multiple adjustment for differences in skin type and time spent outdoors.

Sunscreens suppress natural warnings of overexposure to the sun and allow excessive exposure to wavelengths of sunlight which they do not block. Because sunscreens create a false sense of security, more effective measures to reduce sunlight exposure, such as limiting time spent in the sun or use of hats and clothing, may be ignored.

It is time to review the efficacy of sunscreens in the prevention of melanoma and basal cell carcinomas. Untested but wide-spread public health recommendations concerning the use of sunscreens for the prevention of skin cancer may be more harmful than advice to control sun exposure by more traditional means. □

Cedric F. Garland, DrPH, FACE
Frank C. Garland, PhD, MACE
Edward D. Gorham, MPH

References

1. Cripps DJ, Hegedus S. Protection factor of sunscreens to monochromatic radiation. *Arch Dermatol.* 1974;7:202–204.
2. Rothman S, Rubin J. Sunburn and para-aminobenzoic acid. *J Invest Dermatol.* 1942;5:445–457.
3. Knox JM, Guin J, Cockerell EG. Benzophenones: ultraviolet light absorbing agents. *J Invest Dermatol.* 1958;30:435–444.
4. Pathak MA, Fitzpatrick TG, Frenk E. Evaluation of topical agents that prevent sunburn—superiority of para-aminobenzoic acid and its ester in ethyl alcohol. *N Engl J Med.* 1969;280:1459–1463.
5. Kligman LH, Akin FJ, Kligman AM. Sunscreens prevent ultraviolet carcinogenesis. *J Am Acad Dermatol.* 1980;3:30–35.
6. American Medical Association, Council on Scientific Affairs. Harmful effects of ultraviolet radiation. *JAMA.* 1989;262:380–384.
7. *Proceedings of the NIH Consensus Conference on Ultraviolet Light and the Skin.* Washington, DC; May 10, 1989.
8. Schulze R, Gräfe K. In: Urbach F, ed. *The Biologic Effects of Ultraviolet Radiation.* New York, NY: Pergamon Press; 1969:359–373.
9. Hurwitz S. The sun and sunscreen protection: recommendations for children. *J Dermatol Surg Oncol.* 1988;14:657–660.
10. Knox JM, Griffin AC, Hakim HE. Protection from ultraviolet carcinogenesis. *J Invest Dermatol.* 1960;34:51–57.
11. Johnson EY, Lookingbill DP. Sunscreen use and sun exposure: trends in a white population. *Arch Dermatol.* 1984;120:727–731.
12. Willis I, Kligman AM. Aminobenzoic acid and its esters: the quest for more effective sunscreens. *Arch Dermatol.* 1970;102:405–417.
13. Lee JAH. The relationship between malignant melanoma of the skin and exposure to sunlight. *Photochem Photobiol.* 1989;50:493–496.
14. Jensen OM, Bolander AM. Trends in malignant melanoma of the skin. *WHO Stat Q.* 1980;33:2–26.
15. Magnus K. Incidence of malignant melanoma of the skin in 5 Nordic countries: significance of solar radiation. *Int J Cancer.* 1977;20:477.
16. Magnus K. Malignant melanoma in Norway. *Tidsskr Nor Laegeforen.* 1986;106:2309–2313.
17. Gallagher RP, Elwood JM, Hill GB. Risk factors for cutaneous malignant melanoma: the Western Canada Melanoma Study. *Rec Res Cancer Res.* 1986;102:38–55.
18. National Center for Health Statistics. *Vital Statistics of the United States: Mortality.* Washington, DC: US Government Printing Office; 1955–1987.
19. Armstrong BK, Howell CM. Trends in mortality from melanoma in Australia. *Med J Austral.* 1987;147:150. Letter.
20. Muir C, Waterhouse J, Mack T, et al., eds. *Cancer Incidence in Five Continents.* Vol 5. Lyon, France: International Agency for Research on Cancer; 1987.
21. US Food and Drug Administration. Sunscreen drug products for over-the-counter use. *Federal Register.* 1978;43:38206–38269.
22. Frederick JE, Lubin D. The budget of biologically active ultraviolet radiation in the earth-atmosphere system. *J Geophys Res.* 1988;93:3825–3832.
23. Kaidbey K, Gange RW. Comparison of methods for assessing photoprotection against ultraviolet A in vivo. *J Am Acad Dermatol.* 1987;16:346–353.
24. Staberg B, Wulf HC, Poulsen T, et al. The carcinogenic effect of sequential artificial sunlight + UVA irradiation in hairless mice: consequences for solarium 'therapy.' *Arch Dermatol.* 1983;119:641–643.
25. Staberg B, Wulf HC, Klemp P, et al. The carcinogenic effect of UVA irradiation. *J Invest Dermatol.* 1983;81:517–519.
26. Kaidbey KH, Agin PP, Sayre RM, Kligman AH. Photoprotection by melanin—a comparison of black and caucasian skin. *J Am Acad Dermatol.* 1979;1:249–260.
27. Sayre RM, Desrochers DL, Wilson CJ, et al. Skin type, minimal erythema dose (MED), and sunlight acclimatization. *J Am Acad Dermatol.* 1981;5:439–443.
28. Matsuoka LY, Freaney R, Meade A, et al. Chronic sunscreen use decreases circulating concentrations of 25-hydroxyvitamin D. *Arch Dermatol.* 1988;124:1802–1804.
29. Eisman JA, Barkla DH, Tutton PJM. Suppression of in vivo growth of human cancer solid tumor xenografts by 1,25-dihydroxyvitamin D_3. *Cancer Res.* 1987;47:21–25.
30. Graham S, Marshall J, Haughey B, et al. An enquiry into the epidemiology of melanoma. *Am J Epidemiol.* 1985;122:606–619.
31. Hunter DJ, Colditz GA, Stampfer MJ, et al. Risk factors for basal cell carcinoma in a prospective cohort of women. *Ann Epidemiol.* 1990;1:13–23.

Requests for reprints should be sent to Cedric F. Garland, DrPH, University of California, San Diego, Department of Community and Family Medicine, Division of Epidemiology, M-007, 9500 Gilman Drive, La Jolla, CA 92093-0607.

Safe Sexual Practices Not Reliably Maintained by Homosexual Men

Since the start of the Amsterdam Cohort Study among homosexual men in October 1984, the annual incidence of HIV infections declined from 8.9% in 1985 to 1.0% in 1989.[1] In 1990, however, HIV incidence rose to 2.8%. To assess whether possible changes in sexual behavior were related to this increase in HIV infection, we compared the 17 men who seroconverted as of December 1989 with all 521 participants who remained seronegative throughout the study period (1984 to 1990). In addition, changes in the probability of infection through anogenital receptive contact per partner were investigated.

We observed that among recent seroconverters the proportion of men practicing anogenital contact (protected or unprotected) increased from 33% in the first half of 1989 to 63% in the first half of 1990. Among seronegative men a significant lower proportion of 30% reported anogenital contact in the first half of 1990 ($\chi^2 = 7.9, P < .01$).

The mean number of partners with whom seroconverted men had anal sex, with or without condom, also increased significantly, from 0.8 in the first half of 1989 to 2.4 in the first half of 1990 (Wilcoxon, $Z = 2.4, P < .02$). This is significantly higher than the mean of 0.6 partners with whom seronegative men reported anogenital contact in the first half of 1990 (Student's $t = 4.4$, $df = 443$, $P < .01$).

No increase, however, was found in the mean number of partners with whom seroconverted participants reported only unprotected anal sex. This implies that the observed increase in the incidence of HIV infection is related to inconsistent or inadequate condom use. Alternatively, participants' reports of unprotected anal sex might have been incomplete.

Only two out of 17 seroconverted men reported not engaging in anogenital contact but engaging in receptive orogen-